EFFECT OF MALE SEX HORMONE ON THE RESISTANCE OF CHICKENS TO PARASITISM

by

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INTRODUCTION AND REVIEW OF LITERATURE

Resistance of animals to metazoan parasites has been a frequent subject of investigation during the last two decades. Some authors have used the term resistance loosely and at times in an ambiguous manner. Others have attempted to define resistance by dividing it into three types; natural, acquired and age-resistance. It is generally accepted that natural resistance may be defined as resistance to original infections. Acquired resistance is resistance that is built up by the host as a result of becoming infected. Age resistance is characterized by a gradual increase of resistance which develops during the growing period of a young animal.

Chandler (1932a) found that in studying resistance two different problems arise, the resistance of the host to further infection and its resistance to the effects of the parasites. Criteria for the measurement of the degree of resistance have been the comparative rate of growth of parasites in hosts of different ages (Herrick, 1926); comparative numbers of parasites that are able to live in hosts, effects of parasites on younger and older hosts (Ackert and Herrick, 1928); resistance of a host to reinfection (Chandler, 1932); and resistance of a host to the development and egg laying ability of worms (Stoll, 1936).

Nutrition and genetic constitution, other factors in resistance, were studied at Kansas State College by Dr. J. E.

Ackert and his students. A study was made of the resistance of chickens to Ascaridia as affected by diets deficient in vitamins B and A (Zimmerman, Vincent and Ackert, 1926; Ackert, Fisher and Zimmerman, 1927; and Ackert, McIlvaine and Crawford, 1931) and they presented experimental evidence, apparently for the first time in this country, that natural resistance of animals to helminthic infection may be lowered by nutritional deficiencies. Further studies on vitamin D and resistance were made by Ackert and Spindler (1929); on vitamin B and resistance, Ackert and Nolf, (1931). Additional research in dietary supplements and resistance was done by Ackert (1927); Ackert and Beach (1933); Ackert and Whitlock (1935); Ackert, Whitlock and Freeman (1940); and Ackert and Riedel (1946). In 1935 Ackert, Eisenbrandt, Wilmoth, Glading and Pratt found significant differences in the natural resistance of breeds of chickens to the large roundworm Ascaridia galli. Heavy breeds and varieties of chickens, White Plymouth Rocks and Barred Plymouth Rocks, had significantly fewer and smaller Ascaridia than did the lighter White Leghorn and White Minorca breeds. The literature on these and other phases of resistance have been reviewed by La Page (1938), Ackert (1942) and others. Only age resistance will be reviewed in this study.

BRIEF REVIEW OF THE LITERATURE ON AGE RESISTANCE

The first record of increased resistance of animals to parasitism, due to age, appears to be that of Looss who in 1911 found that when he fed hookworm larvae to young dogs, the larvae were able to reach maturity; however, when he attempted the same type of experiment on adult dogs, his results were negative. Ransom and Foster (1920) demonstrated age resistance with Ascaris in pigs. The following year the same investigators were able to show the same type of resistance in chickens with the nematode, Syngamus trachea.

In 1926 Herrick proved that chickens become more resistant to the development of the nematode parasite, Ascaridia perspicillum,[1] as they increase in age. Supplementary evidence of this same phenomenon was found by Ackert and Herrick (1928). In their experiments, terminated at the end of three weeks, the percentage of infections in chickens ranged from 85.7 per cent to 100 per cent. However, in experiments terminated at eight to 45 weeks the infections ranged from 33.4 per cent to 66.7 per cent. By infecting young and adult dogs with larvae of Ancylostoma braziliense, Sarles (1929) demonstrated age resistance with the same host and parasite. Herrick (1928) and McCoy (1931) also demonstrated age resistance in dogs to Ancylostoma caninum. Age resistance against Nippostrongylus muris in laboratory rats was shown by Africa (1931). Chandler (1932) was not able to attain as convincing results as those of Africa. Ackert, Porter and Beach (1935) demonstrated a marked increase in the resistance of chickens to the growth of the intestinal nematode, Ascaridia lineata.[1] Using lengths of worms as the criteria for judging resistance, they found that the maximum resistance in chickens is ordinarily reached at the end of 93 days. Riedel (1948) has presented evidence that mice infected when about six weeks of age were much less resistant to Trichinella spiralis than mice infected when more than five months of age. During the same year Sadun presented evidence to show that chickens develop a strong resistance to infections with the nematode, Ascaridia galli, as a result of either a previous single infection or of repeated infections.

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[1] Now known as Ascaridia galli.

Secretions of the endocrine glands have been suggested as playing a role in resistance and a few investigators have studied these possible relationships. Ackert (1924) found that removal of the chicken thymus did not have any effect on the resistance of the chickens to the development of Ascaridia. Jaffe' (1926) demonstrated that suprarenalectomized rats show a lowered resistance to natural infections. Ackert and Otto (1927) found that chickens parasitized with intestinal round worm, Ascaridia lineata,[2] had no significant effect on the size of the thyroid gland. The resistance of the ram to the nematode, Haemonchus contortus, may be broken down during the breeding season according to Stoll (1936). Ackert and his students have found that resistance of chickens to the growth of the nematode, Ascaridia galli, increases until the chickens are about four months of age. As white leghorn chickens are approaching sexual maturity at this age, it seemed that the male hormone might be a factor in the resistance. Therefore, it became of interest to ascertain if injections of male sex hormone would increase the resistance of young cockerels to parasitism.

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[1] Now known as Ascaridia galli.

[2] Now known as Ascaridia galli.

MATERIALS AND METHODS

The animals used in these experiments were single combed white leghorn and hybrid cockerels obtained as day old chicks from commercially approved hatcheries. Upon arrival the chicks were banded and placed in electrically controlled brooders. The chicks were fed on a well balanced standard diet.

The chicks were divided into two groups according to weight so that for every chicken in Group I there was a chicken of about equal weight in Group II. In all the experiments the birds in Group I received the injections of male sex hormone while the birds in Group II were the control group.

At this time the young cockerels were given their first injection of male sex hormone. The chicks were injected with either a 0.1 cubic centimeter or a 0.2 cc dosage of male sex hormone every other day except on Sundays for a period of about three weeks. Ten injections in all were given the chicks. The hormone was administered intramuscularly in the form of the ester, testosterone propionate, suspended in vegetable oil. The administered male hormone had the concentration of 25 milligrams of crystalline testosterone propionate per cc of vegetable oil. Site of the intramuscular injection was the Pectoralis majoris muscle.

Following the initial injection all the cockerels were parasitized with approximate numbers of embryonated, viable Ascaridia galli eggs. The Ascaridia eggs were obtained from the bodies of living worms. Following removal of the two uteri from the worms the eggs were forced out of the uteri into a Petri dish by applying a slight pressure to the wall of the uterus with a spatula. Uterine fragments were removed and the egg mass was covered with distilled water to a depth of four millimeters. Prevention of mold growth was accomplished by adding a few drops of two per cent formalin to the egg culture according to Ackert et al. (1935). The eggs were incubated at 28 degrees Centigrade and examined periodically. When the eggs had reached the coiled embryo stage in their course of development, and molted once in the shell they were then infective and ready to be used to parasitize the young chicks.

All the chicks were parasitized by the drop method of Riedel (1947). This method involves the use of a 50 cc flat stoppered dropping bottle containing about one-half inch of previously washed sand to which was added the viable Ascaridia eggs. Water was then added to bring the solution well above the sand. After shaking the bottle vigorously to separate the masses of eggs so as to form a uniform suspension, the process of standardizing the suspension was undertaken. The drops were standardized by placing a drop of the suspension on a slide and counting the number of eggs per drop. After having counted several drops, the average number of eggs per drop was determined. If the concentration of the suspension was too great, more water was added. The drops were then restandardized and when the correct concentration was attained, the cockerels were parasitized by opening the chick's mouth and allowing the proper number of drops to make a total of 200±10 eggs to fall well back into its mouth. Precaution had to be taken to see that the suspension was shaken briefly before each chicken was parasitized so as to maintain a uniform suspension. Ackert et al. (1931) found no marked differences in sizes of infections resulting from feeding doses of 100-300 Ascaridia eggs. For this reason the chickens in these experiments were given dosages well within the above limits.

By separating some of the cervical vertebrae of the neck, the chickens were killed. The digestive tract from the gizzard to the yolk sac diverticulum was removed immediately and the contents, including the worms, were flushed into glass jars by the hydraulic method of Ackert and Nolf (1929). To prevent bacterial action on the worms, a small volume of formalin was added.

The lengths of the worms of each chicken were obtained by making a pencil tracing of the projected images. The worms' images were magnified six times by use of a lens in a photographic bellows. The shadows of the worms were traced with a pencil on thin paper and measured with a milled wheel so calibrated as to reduce the length by six times into millimeters. Comparative numbers and lengths of the worms in each group were used in judging the degree of resistance of the fowl.

Experiment 1

The chicks used in this experiment were white leghorn cockerels. When 39 days of age the cockerels were weighed and separated into two groups of approximately the same weights. Four days later the chickens were parasitized with 100±10 embryonated *Ascaridia galli* eggs. On this same day the birds in Group I received the first injection of male sex hormone. After the initial injection, the chickens were given three a week, totaling 10 in all. Each injection consisted of 0.2 cc of a 25 mg per cc concentration of testosterone propionate.

The male sex hormone began to show its effects on the fowls in Group I about the third day after the initial injection. At the end of the first week of injections the combs of the injected group (Group I) were noticeably larger and of deeper red color than were those of the uninjected group (Group II). The size and intensity of the red colored combs increased constantly during the entire period of experimentation. On about the tenth day following the initial injection several of the cockerels in Group I (injected group) attempted to crow. A week later all of the cockerels in the injected group were crowing in high falsetto voices. During the last week of the experiment it was found that the skin and muscles of the young cockerels were resistant to the puncture of a hypodermic needle. Herrick (1946) found that capons and female fowls given testosterone propionate developed skin and muscle tissue that had significantly greater tensile strength than normal untreated fowls. The behavior of the cockerels in Group I (injected group) was pugnacious at times during the experiment.

At the close of three weeks of parasitism the chickens of this experiment were killed and their intestines were flushed for worms. Group I made a gain in weight of 293 gm as compared to the gain of 381 gm for Group II. (Table 1, Fig. 1).

Although the chickens grew normally in this experiment the degrees of infection do not appear to be normal. More than half of the chickens had no worms at all and those that were infected had only one, two or three worms with the exception of one fowl in Group I which had 12 Ascaridia. Group I had an average of 1.5 worms per bird while Group II had an average of 0.45 worms per bird. The heaviest infection occurred in a chicken in Group I that was well over the final average group weight.

The average length of worms in Group I was 20.7 mm with a range of 4.2 mm to 33.3 mm. In Group II the average length of

Table 1. Comparison of chicken weights, and number and lengths of worms in the chickens of Groups I and II in Experiment 1.

Group I. Hormone injected group					Group II. Control group				
Chick number	Chick's weight in grams		Worms		Chick number	Chick's weight in grams		Worms	
	Initial	Final	Number	Average length (mm)		Initial	Final	Number	Average length (mm)
A937	280	500	0	0.0	A956	316	620	1	14.7
A938	260	520	1	11.2	A957	340	710	0	0.0
A939	308	620	12	22.9	A958	308	640	0	0.0
A940	308	580	1	25.8	A959	320	600	0	0.0
A941	324	560	0	0.0	A960	268	580	0	0.0
A942	280	520	3	14.1	A962	340	650	0	0.0
A943	292	640	0	0.0	A963	260	520	0	0.0
A944	360	680	1	4.2	A964	312	600	1	13.0
A945	304	660	1	15.0	A965	260	500	0	0.0
A946	340	680	3	22.6	A966	316	560	0	0.0
A947	300	640	0	0.0	A967	300	620	2	16.7
A948	240	480	0	0.0	A968	380	720	0	0.0
A949	308	660	1	4.5	A969	260	500	1	13.5
A950	304	610	0	0.0	A970	288	700	0	0.0
A951	300	600	3	14.1	A971	312	636	0	0.0
A952	316	dead	0	0.0	A972	340	760	0	0.0
A953	274	520	2	14.9	A973	272	590	1	21.1
A954	348	730	0	0.0	A974	328	680	3	19.5
A955	320	620	0	0.0	A975	268	590	0	0.0
A961	244	460	3	18.5	A976	328	760	0	0.0
Average	300.5	593.6	1.6	20.7	Average	305.0	626.8	0.45	17.1

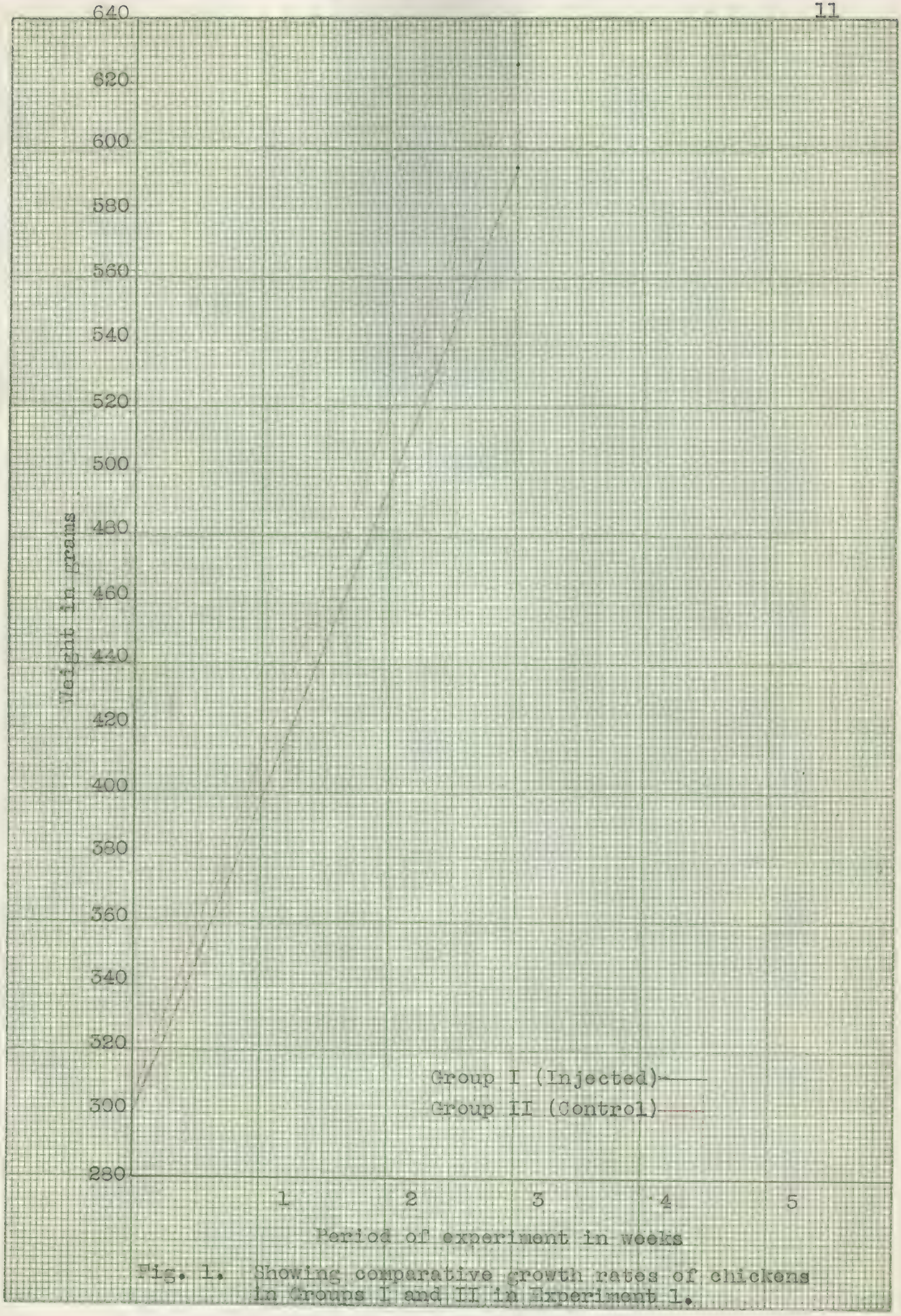

Fig. 1. Showing comparative growth rates of chickens in Groups I and II in Experiment 1.

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worms was 17.1 mm, the range being 13.0 mm to 21.1 mm.

In summarizing the results of this experiment it should be noted that the birds in the injected group made an average of 23 gm less gain in weight than the control group. The injected group also had a larger number of worms which were slightly longer (Table 1).

Experiment 2

The single comb, hybrid cockerels used in this experiment were banded and separated into two groups when they were two weeks old. Each group consisted of 20 birds. When 16 days of age the chicks were parasitized with 200±10 embryonated *Ascaridia galli* eggs. The next day the injections of the testosterone propionate were begun. The amount of hormone injected each time was 0.2 cc of 25 mg per cc concentration. The injections were made intramuscularly three times each week for a period of about three weeks. In all, 10 injections were given to each chicken in the three-week period. The cockerels in Group I were the only birds that received injections of the hormone. All the chicks were kept in compartments of one large cage and the chicks in the control group were caught and handled every time that the birds of the injected group were handled. At the end of three weeks of parasitism the chickens were killed and the worms of each chicken were counted and measured.

In this experiment the control group's gain in weight was more than twice the gain of the injected group, the former making a gain of 63 gm with the latter making a gain of 26 gm (Table 2, Fig. 2).

Group I had a total of 345 worms as compared to 97 worms in the control group. The infestations ranged from one to 60 worms in Group I, while the infections in Group II were from one to 40 worms. The average number of worms per group was 17.3 and 4.9 for Groups I and II, respectively. Each group had three chickens without Ascaridia.

The average length of worms for Group I was 17.0 mm with a range of 20.6 mm to 31.6 mm. In Group II the average worm length was 17.1 mm with a range of 3.2 mm to 22.6 mm.

In regard to the number of worms, Group I with an average of 17.3 worms per bird was much less resistant than Group II with an average of 4.0 worms per bird. With respect to worm lengths Group I with an average length of 17.3 mm also appeared to be less resistant than Group II which had an average worm length of 15.4 mm.

Experiment 3

Single comb, white leghorn cockerels were used in this experiment. When 13 days of age the chicks were separated into two groups of 22 birds each and banded and weighed. On the same day the birds in Group I were given an initial injection of 0.2 cc of a 25 mg per cc concentration of testosterone

Table 2. Comparison of chicken weights, and number and lengths of worms in the chickens of Groups I and II in Experiment 2.

Group I. Cortone injected group					Group II. Control group				
Chick number	Chick's weight in grams		Worms		Chick number	Chick's weight in grams		Worms	
	Initial	Final	Number	Average length (mm)		Initial	Final	Number	Average length (mm)
A992	92	210	5	12.6	A1017	98	270	1	10.0
A993	88	180	50	16.8	A1018	104	200	1	3.6
A994	90	214	0	0.0	A1020	96	242	0	0.0
A995	96	212	1	30.8	A1021	90	210	3	11.7
A996	96	240	38	12.1	A1022	102	230	3	6.7
A997	108	224	1	3.5	A1024	88	230	1	0.7
A1000	92	242	60	20.6	A1025	92	214	4	4.2
A1001	96	170	52	13.4	A1026	88	224	1	3.2
A1002	94	231	0	----	A1028	90	238	1	3.5
A1003	88	190	3	10.7	A1029	90	270	40	15.0
A1004	88	190	4	6.7	A1030	82	150	0	0.0
A1006	90	220	26	15.2	A1031	82	270	3	16.0
A1007	88	198	6	15.5	A1032	92	240	0	0.0
A1010	88	192	10	8.6	A1033	98	238	1	4.4
A1011	130	222	2	12.9	A1034	102	230	3	4.6
A1012	88	210	18	17.0	A1035	96	270	1	10.6
A1013	100	240	39	17.2	A1036	86	250	32	22.6
A1014	90	220	0	0.0	A1037	88	200	1	3.2
A1015	108	200	0	0.0	A1040	90	200	1	5.6
A1016	80	220	19	15.9	A1041	94	Died	-	-----
Average	93.6	221.8	17.3	17.8	Average	97.0	254.4	4.9	15.4

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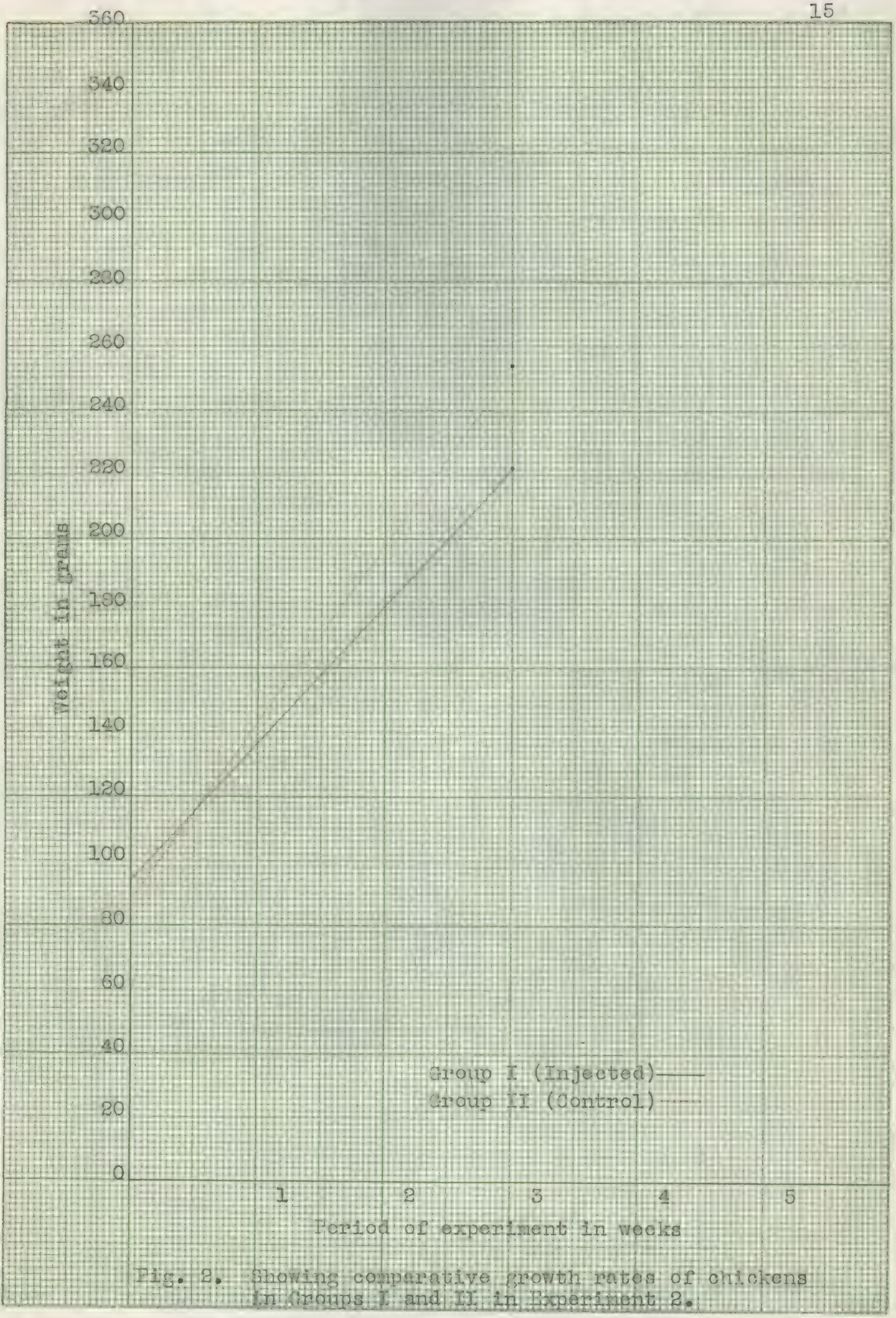

Fig. 2. Showing comparative growth rates of chickens in Groups I and II in Experiment 2.

propionate. Three days later the same group was given another injection of 0.1 cc of a 25 mg per cc concentration of male sex hormone. Every subsequent injection was of the same concentration as the latter dosage. The injections were given at the same time interval as in the previous experiments. At the time of this second injection all the cockerels were parasitized with 400±10 embryonated _Ascaridia galli_ eggs.

In this experiment Group I made a gain in weight of 231 gm as compared to the gain of 305 gm in Group II (Table 3). As can be seen by Fig. 3, the control group made its largest weekly gain during the final week of the experiment.

The number of worms in Group I was eight and a half times the number in Group II while the average number of worms per bird was 6.4 and 0.82, respectively. The heaviest infection of 39 worms in Group I was in a chicken 184 gm below the average group weight; the bird with the lightest infection of one worm was 11 gm above the average group weight. In Group II the heaviest infection of three worms was in two birds, one well above and one slightly below the average group weight.

To summarize the results of this experiment it can be said that Group I was less resistant to the metazoan infection than Group II, judging from the numbers of worms in each respective group.

Table 3. Comparison of chicken weights, and number of worms in the chickens of Groups I and II in Experiment 3.

Group I. Hormone injected group				Group II. Control group			
Chick number	Chick's weight in grams		Worms	Chick number	Chick's weight in grams		Worms
	Initial	Final	Number		Initial	Final	Number
A2404	146	486	0	A2426	154	432	0
A2405	147	424	12	A2427	140	460	0
A2406	122	364	0	A2428	134	485	0
A2407	155	330	32	A2429	156	500	0
A2408	145	308	17	A2430	128	376	0
A2409	175	474	lost	A2431	132	----	-
A2410	131	320	0	A2432	153	440	1
A2411	148	415	0	A2433	109	344	-
A2412	126	392	4	A2434	143	468	-
A2413	150	464	-	A2435	118	400	1
A2414	152	240	30	A2436	110	430	2
A2415	143	290	3	A2437	152	475	0
A2416	142	400	0	A2438	144	478	1
A2417	129	356	0	A2439	157	480	-
A2418	130	350	-	A2440	132	438	-
A2419	158	364	lost	A2441	153	443	3
A2420	147	324	9	A2442	132	378	0
A2421	150	436	0	A2443	123	425	3
A2422	154	326	0	A2444	129	416	2
A2423	157	444	lost	A2445	128	438	0
A2424	126	385	1	A2446	165	504	1
A2425	146	400	1	A2447	114	390	0
Average	143	374	6.4	Average	135	441	0.82

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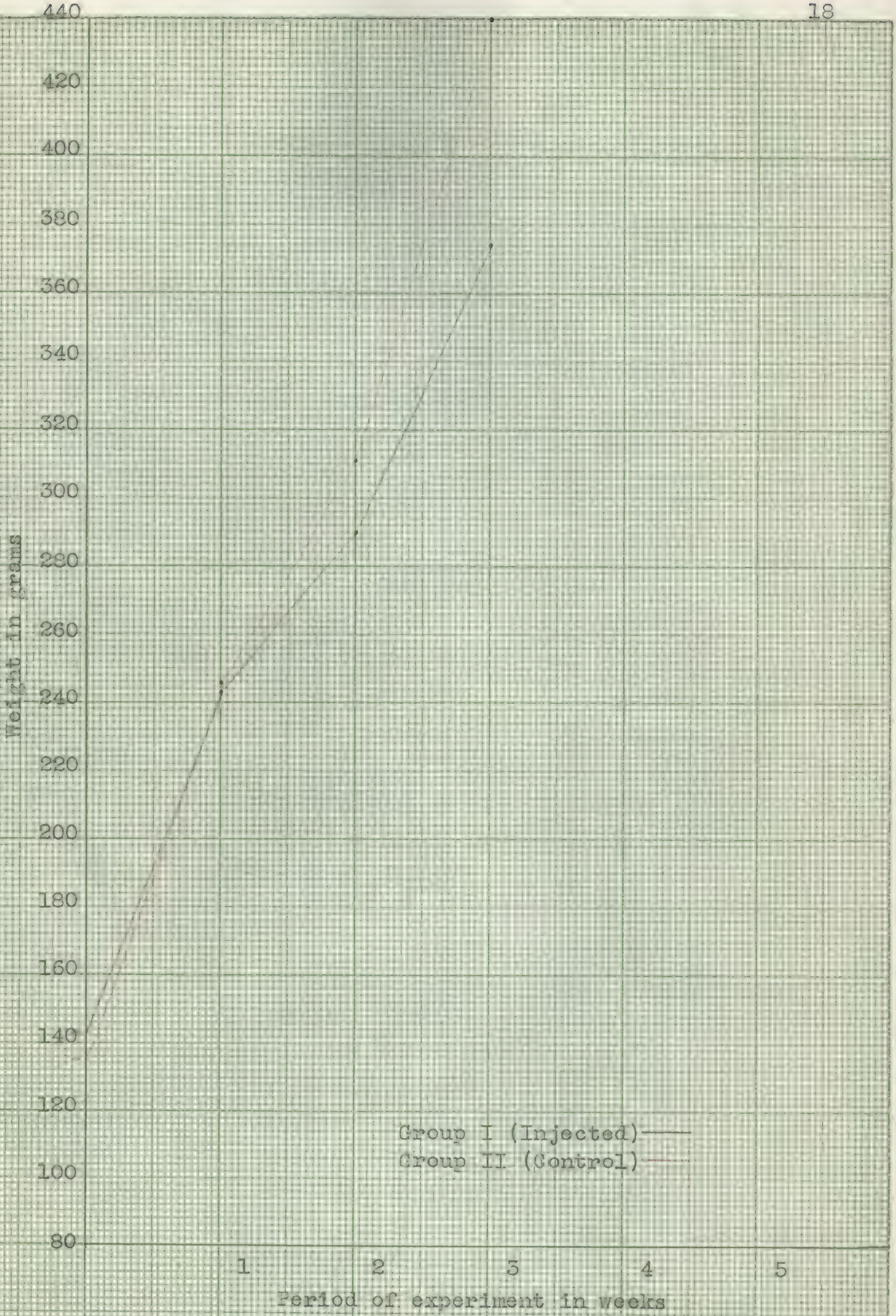

Fig. 3. Showing comparative growth rates of chickens in Groups I and II in Experiment 3.

Experiment 4

The single comb, white leghorn cockerels used in this experiment were received as day old birds and banded upon the day of arrival. Ten days later the chickens were weighed and separated into two groups according to weight. Each group consisted of 25 birds. At this same time the injection of male sex hormone was started. The cockerels in Group I were injected with male sex hormone every other day except Sunday at the rate of 0.1 cc (25 mg) per injection. In an attempt to eliminate any error in the weights of the young cockerels, the control group was also injected with corresponding amounts of pure vegetable oil. It was thought that increased handling of the chicks in the injected group, might affect their growth and resistance. After 14 days the chickens were parasitized with 200±10 embryonated *Ascaridia galli* eggs; and on the 21st day after parasitism they were killed, and the worms of each chicken were counted and measured.

The initial average weekly gains in weight of the two groups were approximately the same (Table 4, Fig. 4). However, at the end of a two-week period the average weekly gain in weight of Group II began to gather momentum and at the end of the fifth week, it had completely eclipsed the average weekly weight gain of Group I. Failure of the chickens in Group I to match the weekly gains in weight of the control group was indicative of a higher degree of infection caused by a decrease

Table 4. Comparison of chicken weights, and number and lengths of worms in the chickens of Groups I and II in Experiment 4.

Group I. Hormone injected group					Group II. Control group				
Chick number	Chick's weight in grams		Worms		Chick number	Chick's weight in grams		Worms	
	Initial	Final	Number	Average length (mm)		Initial	Final	Number	Average length (mm)
A3055	97	432	0	.0	A3073	84	410	10	20.6
A3056	84	307	11	22.9	A3080	80	436	32	22.5
A3057	70	330	2	14.0	A3081	80	356	5	19.7
A3058	87	330	6	15.3	A3082	75	294	34	21.0
A3059	70	287	10	25.4	A3083	86	392	23	25.3
A3060	81	350	0	23.5	A3084	77	404	0	.0
A3061	88	320	9	19.9	A3085	85	308	0	.0
A3062	88	----	3	16.0	A3086	73	320	0	.0
A3063	72	265	47	20.2	A3087	84	434	1	19.6
A3064	76	290	32	18.2	A3088	80	308	0	.0
A3065	94	213	21	18.5	A3089	87	420	0	.0
A3066	75	----	15	26.7	A3090	80	368	21	21.0
A3067	82	385	3	20.8	A3091	75	290	6	22.3
A3068	85	280	4	10.3	A3092	75	302	5	15.5
A3069	80	332	3	19.0	A3093	88	402	2	19.5
A3070	89	340	14	19.3	A3094	88	300	0	.0
A3071	80	320	4	20.9	A3095	84	378	0	.0
A3072	96	404	20	25.2	A3097	84	456	0	.0
A3074	62	298	34	26.9	A3098	70	262	0	.0
A3075	83	264	9	18.0	A3099	75	382	0	.0
A3076	74	234	0	.0	A3100	84	442	3	22.7
A3077	76	264	22	20.7	A3101	84	440	0	.0
A3078	90	330	4	16.9	A3102	83	364	2	20.6
A3079	85	348	17	20.3	A3104	76	320	15	18.9
A3096	72	382	1	19.0	A3105	84	396	66	22.3
A3103	76	286	13	25.0					
Average	82.3	291.2	12.2	26.2	Average	82.9	373.8	9.1	12.0

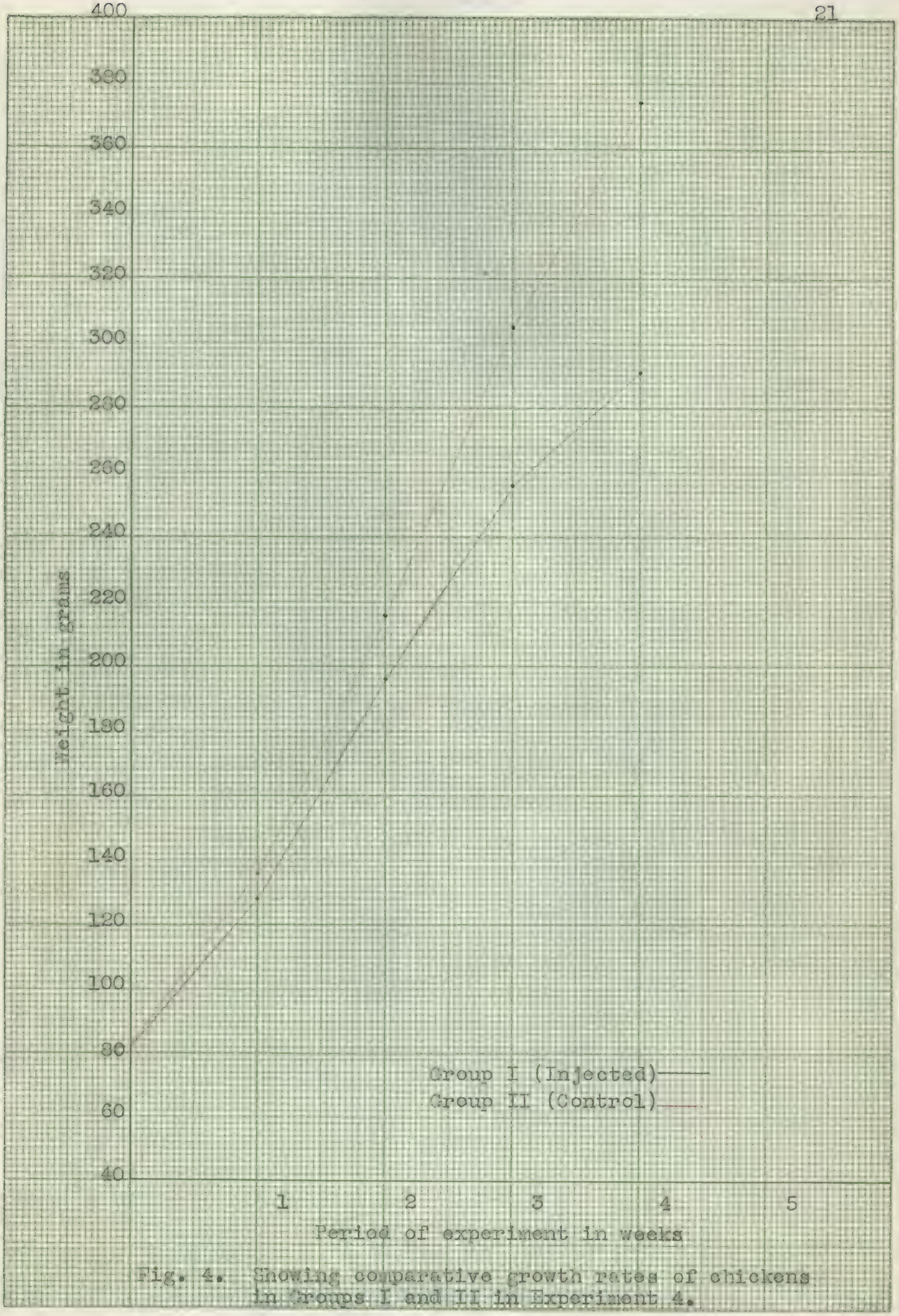

Fig. 4. Showing comparative growth rates of chickens in Groups I and II in Experiment 4.

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in their resistance. Ackert and Herrick (1928) demonstrated that the loss of weight in young chickens can be correlated with the degree of parasitism.

Concerning the numbers of worms, Group I had 310 worms taken from 24 of the 26 chickens: Group II had 228 worms taken from 14 of the 25 chickens. The infections ranged from one to 47 worms per bird in Group I, as compared to the range of one to 66 worms per bird in Group II. The average number of worms per bird for Group I was 12.2 worms and Group II, 9.1 worms.

The heaviest infection of 47 worms in Group I was in a chicken 26 gm below the average group weight while the smallest infection of one worm was in a bird that weighed 31 gm more than the average group weight. Three other birds in Group I with small infections of two or three worms, weighed at least 35 gm more than the average group weight. In Group II the heaviest infections of 66 worms and 34 worms, respectively, were in two chickens, one slightly above and one well below the final average group weight. Only one chicken of Group II had but one worm and its weight was 60 gm above the average weight for the group.

The average length of worms for Group I was 26.2 mm with a range of 10.5 mm to 28.9 mm. In Group II the average worm length was 12.8 mm with a range of 13.8 mm to 25.3 mm.

Using numbers and lengths of worms as criteria for determining the degree of resistance of the chickens, it appears that Group I with an average of 12.2 worms per chicken was less resistant than was Group II with an average of 9.1 worms per chicken. The data from lengths of worms indicate that the average worm length was twice as much in Group I, the injected group, as in Group II, the control group; therefore, the chickens in Group I were markedly more susceptible to the worms than the chickens in Group II.

Experiment 5

In this experiment the chicks were banded and grouped according to weight when 10 days old with 20 birds in each group. At this time the birds in Group I were given the first injection of male sex hormone. The group was given 0.1 cc of a 25 mg concentration of testosterone propionate every other day except Sunday for a period of three weeks. The birds received a total of 10 injections. On the second day following the initial injection, the cockerels in Group I and II were parasitized with 200±10 viable *Ascaridia galli* eggs. Twenty-eight day later, the chicks in both groups were killed and the worms removed.

Group II made a larger gain in weight than Group I; Group II gained 270 gm while Group I gained 245 gm (Table 5, Fig. 5).

Groups I and II each had two fowls without *Ascaridia*. The range of infections from the lowest to the highest of Group I was three to 52 worms per bird; Group II ranged from one to 64 worms per bird. In contrast to the average of 10.8 worms per

Table 5. Comparison of chicken weights, and number and lengths of worms in the chickens of Groups I and II in Experiment 5.

Group I. Hormone injected group					Group II. Control group				
Chick number	Chick's weight in grams		Worms		Chick number	Chick's weight in grams		Worms	
	Initial	Final	Number	Average length (mm)		Initial	Final	Number	Average length (mm)
A3257	71	312	0	00.0	A3277	92	424	36	41.4
A3258	74	270	42	44.6	A3278	90	424	20	30.3
A3259	90	298	9	38.9	A3279	84	346	83	43.4
A3260	92	192	45	45.9	A3280	78	336	0	00.0
A3261	89	378	16	42.0	A3281	88	432	68	30.1
A3262	93	403	30	42.4	A3282	83	340	44	45.0
A3263	85	364	40	43.2	A3283	84	402	39	30.2
A3264	94	290	3	34.7	A3284	90	334	94	36.8
A3265	102	402	8	27.0	A3285	86	466	47	30.5
A3266	86	268	6	43.7	A3286	84	370	19	31.0
A3267	79	285	30	44.0	A3287	85	344	1	36.0
A3268	99	410	12	40.7	A3288	86	360	9	43.1
A3269	88	336	9	30.2	A3289	90	370	65	38.7
A3270	84	312	7	43.7	A3290	76	302	36	46.9
A3271	92	316	52	41.3	A3291	84	365	40	43.8
A3272	76	334	0	00.0	A3292	86	300	0	00.0
A3273	92	371	17	22.8	A3293	84	336	20	30.4
A3274	86	326	5	40.2	A3294	78	328	60	49.4
A3275	94	346	3	40.4	A3295	81	340	7	34.3
A3276	94	340	48	35.2	A3296	82	344	41	30.8
Average	86.0	329.6	19.8	40.6	Average	83.0	363.1	32.3	40.7

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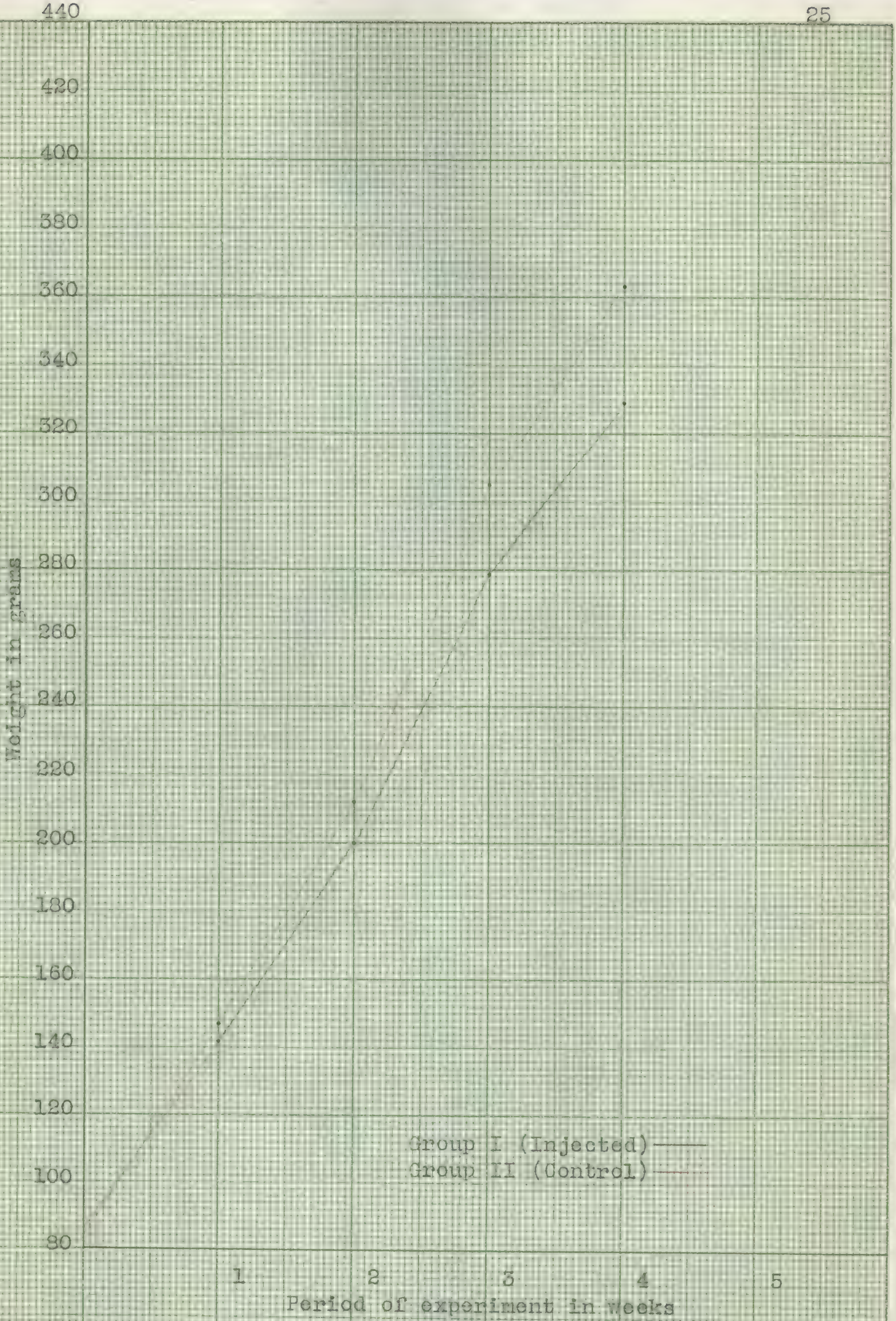

Fig. 5. Showing comparative growth rates of chickens in Groups I and II in Experiment 5.

bird in the injected group, the control group had an average of 32.8 worms per bird.

The heaviest infection of 52 worms in Group I was in a bird below the average group weight. The lowest infection was in a chicken slightly below the average group weight. In Group II a bird whose weight was 130 gm below the average group weight had the heaviest infection of 84 worms. The smallest infection of one worm was in a bird whose weight was 19 gm below the average group weight.

The shortest worms of Group I averaged 22.0 mm and were taken from a chicken whose weight was well above the average group weight. The longest worms averaging 48.2 mm were taken from a chicken whose weight was well above the average group weight. In Group II the shortest average worm length of 30.3 mm was taken from a bird far above the average group weight. The longest worms, averaging 45.9 mm, for the group were taken from two chickens, one of average group weight and the other well below. The average worm length for Group I was 40.6 mm, as compared to 40.7 mm in Group II.

Using the lengths of worms as a measure of resistance, Table 5 indicates that the chickens in the injected group and the control group are equally susceptible to the nemathelminth. Basing resistance on the numbers of worms per group, Group I was markedly more resistant than Group II. This is the first instance in the series of five experiments in which the controls (Group II) had more worms than did the injected chickens (Group

I).

Table 6. Summary of average worm numbers and average worm lengths.

Experiment	Group I		Group II	
	Number per chicken (worms)	Length per chicken (worms, mm)	Number per chicken (worms)	Length per chicken (worms, mm)
1	1.6	20.7	0.45	17.1
2	17.5	17.8	4.90	15.4
3	6.4	———	0.22	———
4	12.2	26.2	0.10	12.8
5	19.8	40.6	42.30	40.7
Average	11.5	26.3	9.60	21.5

COMBINED RESULTS

In reviewing the combined results of the five experiments Group I (injected group) had an average of 11.5 worms per chicken while Group II (control group) had an average of 9.6 worms per fowl. For Groups I and II the average lengths of worms were 26.3 mm and 21.5 mm, respectively.

The nematodes in four of the five experiments were longer in Group I than in Group II, except in Experiment 5 (Table 6), where the average worm lengths were essentially equal. In the combined results the worms in Group I averaged 4.8 mm longer than those of Group II. The results given in Table 6 show that there is a definite trend toward lowered resistance in Group I (injected group).

In respect to worm numbers, the results of four of the five experiments show that the chickens in Group I were more susceptible to the nematode infections. In Experiment 6 however, the control fowls had more worms. This probably was due to the Group II chickens receiving more viable eggs (administered by the drop method) than did the Group I chickens. Comparison of growth of worms (average length) did not indicate that Group II was more susceptible (Table 6).

Using worm numbers and worm lengths as the criteria for judging resistance, the combined data indicate that young male chickens injected intramuscularly with testosterone propionate are less resistant to the fowl nematode, _Ascaridia galli_, than are male chickens which have not received the male hormone injections.

DISCUSSION

At the termination of each individual experiment, the greatest visible difference between the two groups of cockerels was in the size of combs and wattles. The comb measurements of a fowl in Group I (injected) measured 77 mm in length and 47 mm in height as compared to 43 mm and 22 mm of a fowl's comb in Group II. Both birds were selected as being representative of their respective groups. In Experiment 5 the combs and wattles were removed from all the fowls in Group I and Group II and weighed. The combined weight of the combs and wattles taken from Group I (injected) was 90 gm while those of Group II (control) weighed 42 gm.

Group I (injected) consistently failed to match the gains in weight of Group II (control). At first it was assumed that this might be due partially to the fact that the chickens in Group I (injected group), were handled more frequently. However, this assumption proved to be unwarranted because in experiments in which both groups of chickens were handled the same amount, and in other experiments where the chickens in Group I were subject to more frequent handling, the gains in weight were essentially the same. Explanation of this difference in weight gains may be conceivably attributed to the tri-weekly injections of the foreign body substance, testosterone propionate, which may have interfered with the normal physiological functions including the deposition of the growth inhibiting factor in the goblet cell mucin (Ackert, Edgar and Frick, 1939; Frick and Ackert, 1948).

No correlation can be made between the weights of chickens and worm numbers or weight of chickens and worm lengths. Combined results show that the worms of Group I are more numerous and longer than those of Group II. The worms of Group I were probably longer than those of Group II because the number of infections were higher. Ackert and Herrick (1928) found that chickens with a larger number of infections may have a reduced resistance due to the amount of toxins secreted as waste by the worms. The chickens in Group II probably developed a higher resistance than the chickens in Group I because their

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growth was not retarded as indicated by their more rapid increase in weight. Accompanying growth in immature chickens there is increased production in number of goblet cells which secrete mucin containing an inhibitory worm growth factor (Ackert, Edgar and Frick, 1939; Ackert, Porter and Beach, 1935; Frick and Ackert, 1948).

SUMMARY

1. A series of five experiments was performed on 107 cockerels to ascertain if male sex hormone is a possible factor in the resistance of chickens to the nematode *Ascaridia galli*.

2. The cockerels of each experiment were divided into two equal groups by weight. The chickens in Group I were given tri-weekly injections of testosterone propionate for a period of three weeks.

3. Weekly records of the chicks' weights were made after the chickens were segregated into groups.

4. Chickens in both groups were parasitized with 200 ± 10 embryonated *Ascaridia galli* eggs. Three weeks later the chickens were killed and the intestine removed for flushing of the worms.

5. Group II in each experiment made the largest gain in weight.

6. Criteria for judging the degree of resistance of each group of chickens were the worm numbers and worm lengths.

7. Group I had an average of 11.5 worms per chicken as compared to 9.6 worms per chicken in Group II.

8. The average worm length for Group I was 26.5 mm and for Group II 21.5 mm.

9. Using worm numbers and worm lengths as the criteria for judging resistance, it was found that young male chickens' resistance to the fowl nematode Ascaridia galli was reduced as a result of the intramuscular injections of testosterone propionate.

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